HARVARD SCHOOL OF PUBLIC HEALTH

Department of Environmental Health
Environmental Epidemiology Program

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Hearing on H.R. 881, a bill to ban smoking in all Federal Buildings by the Committee on Public Works and Transportation, Subcommittee on Public Buildings and Grounds.

Tobacco smoke is a known cause of lung cancer. If tobacco smoke were held to the same standards applied to other environmental carcinogens found in public buildings, there is no question it would be banned. We consider it unacceptable to passively expose people in public buildings to asbestos, formaldehyde, or radon. It therefore should not be acceptable to passively expose people in public buildings to tobacco smoke.

The carcinogeniticity of tobacco smoke has been demonstrated by all methods used to assess risk, that is in animal bioassay studies, genotoxicity studies, and epidemiologic studies. Tobacco smoke is not only a recognized cause of cancer, it is a strong carcinogen. Epidemiologic studies have shown that active smokers develop lung cancer at a rate at least ten times that of never smokers. Epidemiologic studies have shown that the risk of lung cancer associated with tobacco smoke increases with exposure, either as measured by number of cigarettes smoked per day, or years of cigarette smoking. There is no evidence from these studies of active smokers that even the smallest exposures to active smoking are free of risk. It follows immediately that exposures to low concentrations of tobacco smoke should be associated with increased risk of lung cancer. **The evidence that tobacco smoke is such a strong cause of lung cancer, without any other consideration, is sufficient to define environmental tobacco smoke as a lung cancer hazard and to ban smoking from all Federal Buildings.**

There is also substantial evidence showing that nonsmokers are passively exposed to tobacco smoke at nontrivial levels. A considerable mass of data has been developed estimating environmental exposures to tobacco smoke, direct measurements of ambient indoor concentrations of tobacco smoke, and direct measures of dose based on biologic tissue samples. Non-smokers have levels of cotinine, a specific nicotine metabolite, which are 1/10% to 2% of those of smokers. **The evidence that large numbers of people in the general population are exposed to this carcinogen is sufficient to define environmental tobacco smoke as a lung cancer hazard and to ban smoking from all Federal Buildings.**

Given that tobacco smoke is associated with increased incidence of lung cancer down to the lowest exposures among active smokers, and that there is widespread environmental exposure to tobacco smoke among nonsmokers, increased lung cancer incidence should be expected among never smokers chronically exposed to environmental tobacco smoke. Indeed, increased incidence of lung cancer has been consistently observed in studies of never smoking women married to husbands who smoke. **The evidence from epidemiologic studies that never smoking women with smoking spouses have increased risk of lung cancer only confirms what was apparent from the carcinogenicity of tobacco smoke itself.**

Given the compelling evidence cited above, the hypothesis to be tested (that is, the null hypothesis) is not whether environmental tobacco smoke has a statistically significant association with lung cancer in these epidemiologic studies. Our prior hypothesis would certainly be that environmental tobacco smoke increases the risk of lung cancer. Thus the hypothesis to be tested is that environmental tobacco smoke does not increase lung cancer risk. Of the 30 studies reviewed by the EPA[1], only one study[2] was not consistent with an increased lung cancer risk associated with spousal smoking. The consistency of these results across so many independent studies showing a positive association between lung cancer and

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[1]Environmental Protection Agency, "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders." EPA/600/6-90/006B, May 1992.

[2]Wu-Williams AH, Samet JH. Environmental tobacco smoke: exposure response relationships in epidemiologic studies. Risk Analysis. 1990:10:1.

spousal smoking is a very strong statement of the robustness of those findings. In epidemiologic studies of effects of environmental hazards such consistency and robustness is a much more important indicator of causality than statistical significance.

The attached figure presents my summary of the available epidemiology data showing the association of lung cancer in non-smoking women with cigarette smoking of their husbands. Results from 32 studies are presented. For each study, the estimated relative odds of lung cancer associated with husband's cigarette smoking is presented along with the 95% Confidence Interval for that estimate. A relative odds greater than one, that is on the right side of the plot, indicates that lung cancer in these nonsmoking women is positively associated with their husbands' smoking. It is clear from this plot that almost all of these studies have found such a positive association.

Critics have suggested that these associations may be the result of bias or confounding in the data. That is, the observed associations are due to some characteristic other than spousel smoking which is related to both lung cancer and spousel smoking. While each study has weaknesses, and bias and confounding must be considered in any study, it is my interpretation of these studies that bias and confounding have generally acted to underestimate the true association between lung cancer and environmental tobacco smoke exposure. Other design issues, such as the fact that spousel smoking is only a crude measure of environmental tobacco smoke exposure in the home, at work, and in other settings, will also produce underestimates of the true association.

Critics have also suggested that the use of meta-analytic techniques, as in the EPA Review, are inappropriate for epidemiologic data. In this I strongly disagree. Meta-analysis is a well developed method for contrasting and combining results from different studies. The concepts and methods are actually a simple extension of statistical analysis commonly used in epidemiology. These methods are commonly used in public health research, particularly

in the evaluation of clinical trials[3]. Meta-analysis has been applied in epidemiologic research for many decades[4] and has recently been reviewed in two scholarly reviews[5,6]. The use of these methods by the EPA is not only appropriate, but is a significant advance in evaluating the epidemiologic literature.

In my opinion, the epidemiologic evidence for an association between lung cancer and environmental tobacco smoke is very compelling. The EPA Review is a comprehensive, rigorous, balanced and scholarly summation of the current state of the science, which supports such a finding. While epidemiologic studies alone cannot demonstrate causality, the universal finding of the carcinogenicity of tobacco smoke in animal and genotoxicity studies corroborates the epidemiology. There is no doubt that tobacco smoke is an environmental carcinogen, which should be banned from Federal Buildings and all other public facilities.

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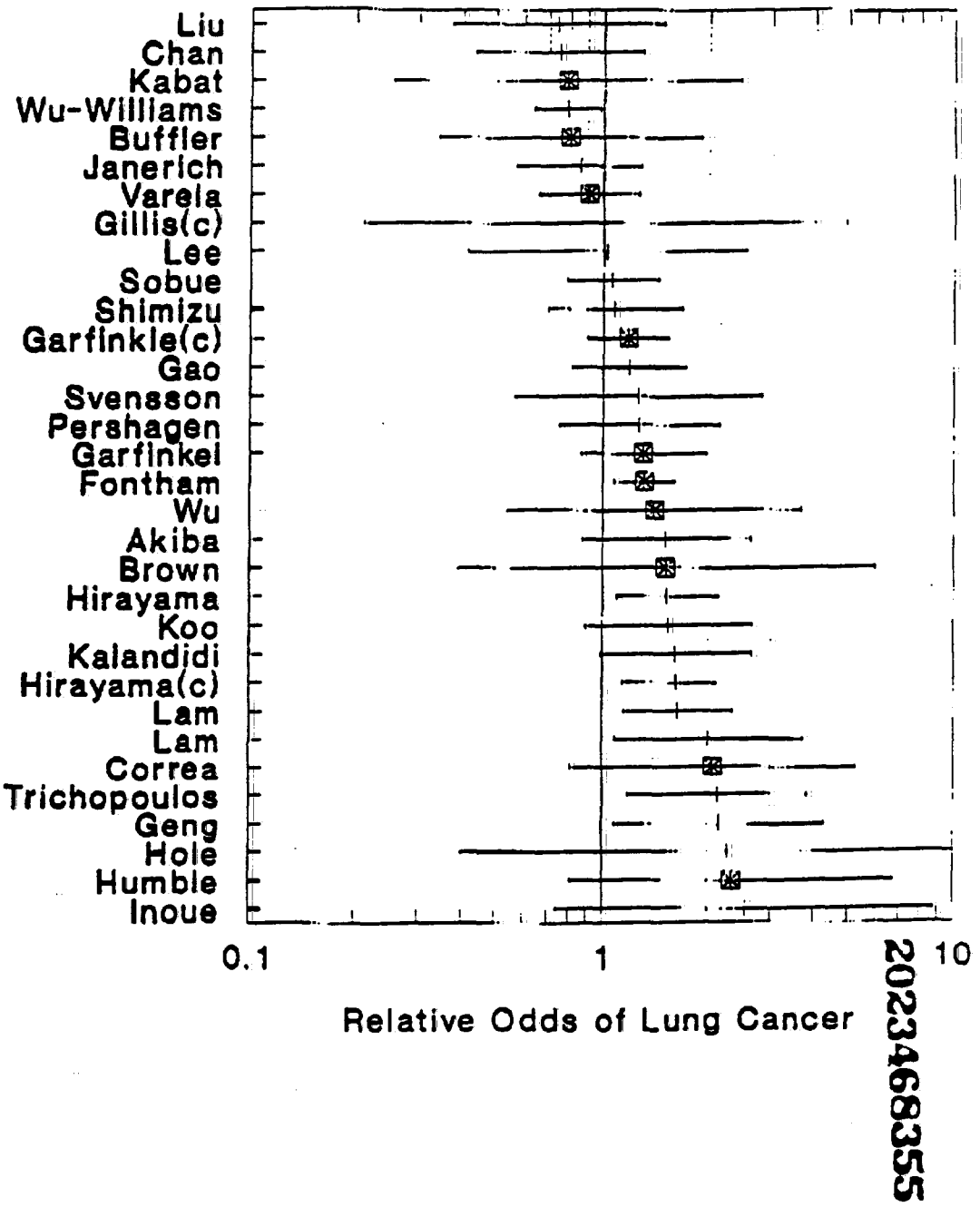
[3]Louis TA, Fineberg HV, Mosteller F. Findings for public health from meta-analysis. Annual Reviews of Public Health 1985; 6:1-20.

[4]MacMahon B, Hutchinson GB. Prenatal x-ray and childhood cancer: a review. Acta Un Int Cancer 1964; 2:1172-1174.

[5]Greenland S. Quantitative methods in the review of epidemiologic literature. Epidemiologic Reviews 1987; 9:1-30.

[6]Dickersin K, Berlin JA. Meta-analysis: State-of-the-Science. Epidemiologic Reviews 1992; 14:154-176.

EPIDEMIOLOGIC STUDIES OF PASSIVE SMOKING & LUNG CANCER



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